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Public Relations

A FEATURE of the current correspondence under the title " A Legitimate Grievance " has been the frequent reference to lack of public relations work. To some extent those who have dwelt upon this matter seem to be unaware of the amount of such work the Association does already, and some correspondents have even suggested the appointment of a public relations officer. In point of fact, the Association has had either a whole-time or part-time public relations officer for the past five years. As more than 1,000 newspapers and periodicals are published every week, it is not surprising that references to the profession and to the B.V.A. are easily lost in this welter of print, but it may be recalled as an example, that consequent upon the last two Congresses, the newspaper Press gave on each occasion about 1,000 inches of editorial space to the Association's affairs. This is not much less than a total of 10 full pages of an ordinary issue of *The Times*.

To recall these facts is not to suggest that the Association's public relations work could not be improved. It most certainly could; but any such improvement calls for consistent support from members of the profession. In newspapers all over the country letters, paragraphs, and articles appear, often with reference to the work of some of the animal welfare societies, which lead the general public to think that these societies are invariably staffed by veterinary surgeons. In cases where their local clinics are not so staffed we suggest to local practitioners that they should bring such matters to our notice so that they may be corrected. Clearly, it is impossible to read individually the hundreds of journals which are published, and it is in this matter that veterinarians in private practice are asked to play their part.

It should be possible also, without offence to professional ethics, to make more widely known to newspaper readers the skill of our profession. It may be recalled that a few months ago veterinary surgeons succeeded in performing an operation which gave back its sight to a horse. The topic was publicised in almost every newspaper without mentioning the veterinarians by name. Here is a useful field in which, again, members of the profession could assist us. If some notable piece of work comes to their notice, especially concerning a domestic pet or a horse, and if they would give us the particulars, it should not be difficult to make such work known while scrupulously maintaining the anonymity of those who performed it. By such means as these the profession can make its work known, and put forward its just claim upon public esteem.

(Concluded on p. 115, col. 2)

Hypomagnesaemic Tetany in Beef Cattle

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EXPERIMENTS carried out during the past 30 years have shown without any doubt that if calves are given cow's whole milk as their *sole* food for long periods, they develop signs of severe nutritional impairment. These include failure of growth, cachexia, anaemia, acute respiratory distress, muscular weakness, bone abnormalities, neuromuscular irritability and convulsions. Few calves survive for longer than 200 days if they are so restricted in their ration.

The anaemia of the milk-fed calf is due to iron deficiency (Knoop, Krauss, & Washburn, 1935); possibly copper is deficient too. The work of Duncan, Huffman, & Robinson (1935), in pursuance of a suggestion of McCandlish (1923) showed that the convulsions and hyperirritability were associated with a lowered serum content of magnesium. Following this work, it has been customary to ascribe the whole sequence of changes observed in such experiments with whole milk to the sequelae of hypomagnesaemia. Thus, the paper of Moore, Hallman, & Sholl (1938), interpreting their histological findings in Huffman's calves, ascribes them specifically to magnesium deficiency, and the signs of this deficiency have become accepted to be the presence of metastatic calcification of the great vessels and myocardium, hyaline degeneration of skeletal muscle and gross kidney degeneration. Specific experiments using artificial diets simulating milk, and virtually free of magnesium have not, however, confirmed these findings. In calves given such diets and dying in convulsions no nephritis has been found, no pathological calcification of any tissue detected by chemical or histological test, and no indication of hyaline degeneration of muscle cells has been seen or detected biochemically. In these experiments magnesium added to the diet prevented the disease (Blaxter, Rook, & MacDonald, 1954a).

It would appear, therefore, that considerable care is necessary before pathological signs in calves are interpreted as being specifically due to a single nutrient deficiency of whole milk. The present paper is concerned with field experience in the diagnosis of magnesium deficiency in suckling calves, and its clinical and pathological distinction from other known disorders of nutritional origin. It shows also that the enzootic muscular dystrophy of Inverness and the adjoining counties in the North of Scotland is quite distinct from hypomagnesaemic tetany, but that both diseases do occur in the area and are causes of serious loss.

Experimental

Material and Methods

Clinical and biochemical evidence was collected from approximately 25 farms in the immediate vicinity of Inverness, and one in Caithness, during the years 1953 and 1954. A large part of the data was obtained incidental to other investigations (Blaxter & Sharman, 1953a, b). Clinical and *post-mortem* methods are self explanatory; magnesium and other chemical determinations were made by modifications of the methods listed by Godden (1935).

The modes of husbandry on all these beef-rearing farms were very similar to those already described by Sharman (1954). The cows were kept indoors throughout the winter, usually until May, and their calves, born in the first three months of the year, received little or no supplementary feeding, relying entirely on the milk of their dams.

Results

The Widespread Occurrence of Hypomagnesaemia in Inverness

In Fig. 1, frequency distributions of the blood serum magnesium and calcium are given for calves on 20 farms in Inverness in two successive years. These farms were not known at the outset to have any problem associated with hypomagnesaemia and were selected solely on the basis of a herd history of muscular dystrophy (Sharman, 1954). The observations made during the first few days of life showed that the serum magnesium was in general above 2 mg. per 100 ml., and the individual values agreed with the usually accepted normal range of 2 to 2.5 mg. per 100 ml. These values were not maintained. A slow fall in magnesium content occurred during the first two months of life, resulting in a considerable distortion of the frequency distributions. This fall was sufficient in some instances to result in the serum magnesium content being depressed to values below what we accept as a tetany threshold of 0.75 to 0.80 mg. per 100 ml. Serum calcium was not affected in the majority of cases, but all values tended to be high.

The above results show that on the beef-suckling farms of Inverness a large proportion of the calves do not maintain their serum magnesium at what is recognised to be a normal level. More detailed results show that not all farms were affected to the same extent and that the presence of large numbers of animals with a low serum content of magnesium

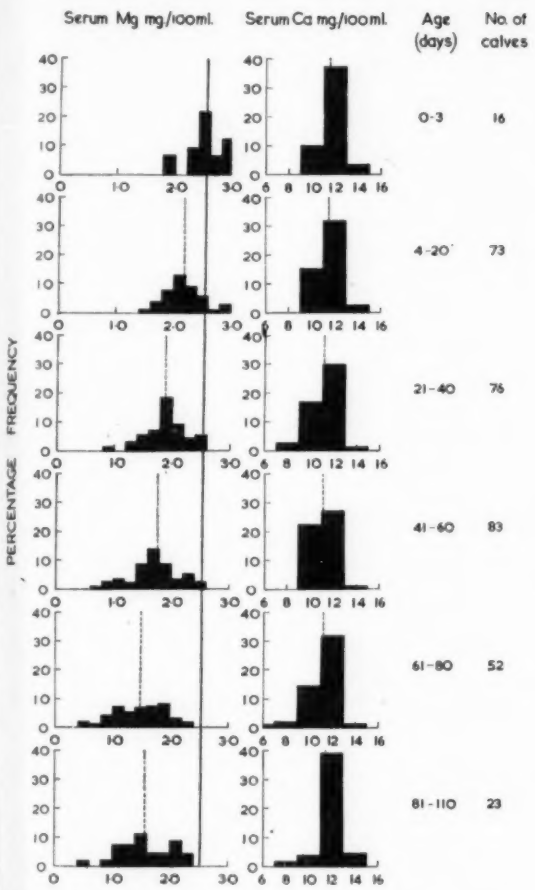

FIG. 1.—Percentage frequency distributions of the serum calcium and magnesium concentration in calves on 20 farms in Inverness in 1953 and 1954, classified according to age of calf.

was not due to any sudden "spontaneous" hypomagnesaemia in a proportion of individuals, but to a slow fall in the concentration of magnesium in the serum of each.

At approximately two weekly intervals in 1953, serum samples were taken from eight calves on each of three of these 20 farms. The mean results are given in Fig. 2. They show that the magnesium content of the serum at birth was not maintained on any of these farms, but that whereas on farm NP, the mean serum level did not fall below 1.9 mg. per 100 ml. during the 74 days of observation, on farm CK and more especially on farm LG, the serum magnesium content of each calf fell considerably, to mean values of 1.6 mg. per 100 ml. and 1.2 mg. per 100 ml. respectively. On the latter farm one individual's blood serum contained only 1.02 mg. per 100 ml. These results, showing a slow fall in the serum magnesium, are similar to those obtained in experiments in which different amounts of magnesium have been given to calves (Blaxter, Rook, & MacDonald, 1954a). In both instances the fall in serum concentration was protracted.

The same three farms were investigated again in 1954, with the results given in Table I. Again, farm NP showed no change in blood serum magnesium concentration, whereas a marked fall in concentration was noted on the other two farms.

These results, together with further less detailed evidence for the remaining 17 farms, suggest that whether or not the serum magnesium is severely depressed, it is dependent in some way on the mode of husbandry on the farms concerned.

TABLE I

MEAN MAGNESIUM CONTENT OF THE SERUM OF CALVES ON THREE FARMS IN TWO SUCCESSIVE YEARS (MG. PER 100 ML.)

Age of calves	Farm NP		Farm LG		Farm CK	
	1953	1954	1953	1954	1953	1954
About 20 days	2·24	2·24	2·07	1·97	2·11	2·19
„ 60 „	2·14	2·14	1·23	1·39	1·60	1·15

On these farms, no signs whatever of any neuromuscular irritability were observed, and, apart from an incidence of muscular dystrophy on farm NP and CK in both 1953 and 1954, no abnormalities were seen in either year.

In no instance on the three farms described above did the blood serum magnesium fall below 1 mg. per 100 ml. in 1953. In the same year, however, an individual calf on one of the 20 farms was found with a blood serum magnesium concentration of less than 0.7 mg. per 100 ml., in which clinical signs of tetany were present. In 1954 the same farm and one further farm of the group had calves showing gross tetany. In no case has tetany been found in calves with a

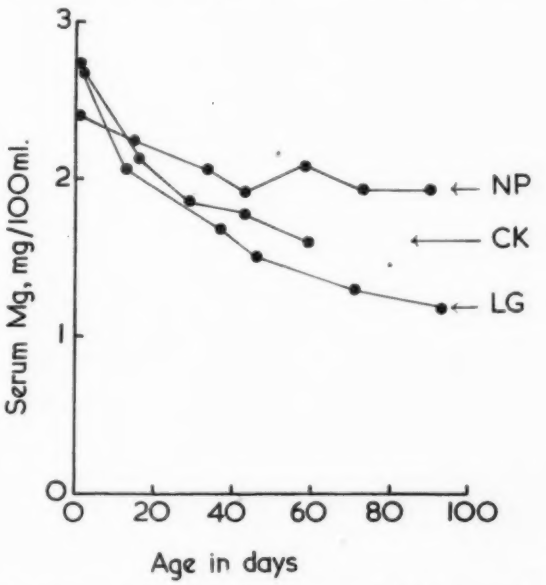

FIG. 2.—The mean magnesium concentration in the serum of calves on three farms in 1953.

serum magnesium concentration greater than 0.8 mg. per 100 ml., but some calves with concentrations less than this have shown few signs. These results agree perfectly with those obtained in experiments with synthetic diets (Blaxter, Rook, & MacDonald, 1954a).

Investigation of the Possible Association with Muscular Dystrophy

The question arose whether the lowered blood serum magnesium had any relationship to enzootic muscular dystrophy, more especially since these farms were selected as "dystrophy prone." In 13 diagnosed cases of muscular dystrophy for which complete biochemical and pathological data were available, the mean serum magnesium, determined approximately one to two weeks prior to its incidence, was 1.72 mg. per 100 ml. In 13 calves of the same age and on the same farms which were given vitamin E and did not develop dystrophy the concentration determined at the same time was 1.74 mg. per 100 ml. The standard error of the difference was ± 0.12. Furthermore, dystrophy occurred on farms where no fall in magnesium occurred at all, that is in calves with a magnesium of the blood serum above 2 mg. per 100 ml.; in fact, the sera of five out of the 13 dystrophic calves contained more than 1.9 mg. per 100 ml. magnesium. The appearance of muscular dystrophy on farm NP (see Tables I and II) makes it abundantly clear that the two diseases are quite distinct.

TABLE II

MAGNESIUM CONTENT OF THE BONES AND THE RATIO OF CALCIUM TO MAGNESIUM IN THE BONE ASH OF BEEF CALVES

Farm reference	Bone	Percentage Mg in ash	Ratio Ca: Mg	Cause of death
NHM 53/2	Cannon	0·67	55:1	Muscular dystrophy
	Rib	0·67	53:1	
NHM 53/1	Cannon	0·53	70:1	Muscular dystrophy
	Rib	0·49	75:1	
AGM 53/2	Cannon	0·61	60:1	Muscular dystrophy
	Rib	0·70	51:1	
NP 54/1	Cannon	0·59	57:1	Muscular dystrophy
	Rib	0·69	53:1	
C 144/54	Cannon	0·50	75:1	Nervous signs and
	Rib	0·46	80:1	muscular dystrophy
C 54 C/1	Cannon	0·38	98:1	Tetanic convulsions
	Rib	0·27	135:1	
C 54 C/2	Cannon	0·36	100:1	Tetanic convulsions
	Rib	0·35	90:1	
C 54 C/3	Cannon	0·32	111:1	Tetanic convulsions
	Rib	0·27	116:1	
C 54 C/108	Cannon	0·30	118:1	Tetanic convulsions
	Rib	0·19	142:1	
Expt. calf H.D.R.I.	Cannon	0·608	54:1	Normal calf, aged
	Rib	0·588	60:1	two months
Expt. calf H.D.R.I.	Cannon	0·660	55:1	Normal calf, aged
	Rib	0·730	50:1	two months
Mg deficient H.D.R.I.	Cannon	0·386	96:1	Mg deficient (acute
	Rib	0·462	92:1	tetany)
Mg deficient H.D.R.I.	Cannon	0·193	175:1	Mg deficient (chronic
	Rib	0·188	185:1	tetany)

It would appear, therefore, that besides being subject to muscular dystrophy, the beef-rearing farms in this area are also prone to a progressive depletion of the magnesium content of the serum, not sufficient in most cases to give rise to clinical signs of tetany, but, on a proportion of farms, capable of causing losses of stock. The relative incidence of these two conditions may be assessed tentatively as 25 per cent. for muscular dystrophy and at least 5 per cent. for hypomagnesaemic tetany. As the detailed reports for the two selected farms show, the condition is a well-defined clinical entity, and usually, but not always, is a problem of the farm young stock as a whole, rather than of a particular animal. In this regard, however, even on a farm with a high incidence of tetany, not all calves show a gross depression of the serum magnesium. In fact, as shown by Farm DC (see below) some calves under identical conditions may have fairly normal serum concentrations. The reasons for this are given later.

Case Reports

Farm LH

This is a mixed arable farm of 120 acres, bordering the sheltered Beauly Firth. A six course rotation of crops is carried out and none of the grass is more than three years old. A 20 per cent. calf mortality was the rule until 1952 when the cause of death was established as muscular dystrophy. In 1953, 75 per cent. of the calves were given α-tocopherol, but no cases of dystrophy or of any abnormality occurred in either the control group or those given the vitamin. No prophylactic treatment with α-tocopherol was given in 1954 to any animal, and when the calves were turned out to grass in mid-March, five calves (calves A, D, R, F, & K) out of the 10 on the farm developed clinical signs of muscular dystrophy, of the skeletal muscle type. These recovered when given α-tocopherol, and three of the remaining calves were given tocopherol as a preventative measure.

No further trouble was experienced with muscular dystrophy, but on May 1st, calf A was found in convulsions. The convulsions had subsided by the time veterinary aid was summoned, but the calf was hyper-excitable. The intravenous injection of 7 grammes magnesium sulphate ≡ 0.7 gramme magnesium) and 9 grammes chloral hydrate abolished hyperexcitability in 10 minutes. Magnesium sulphate was given orally for three days (28 grammes daily, ≡ 2.8 grammes magnesium) and the calf at this time appeared quite normal. Hyperexcitability soon developed again. Convulsions occurred 13 days later, within 18 hours of the commencement of daily dosage with 1 gramme magnesium carbonate. On this occasion the calf lay exhausted for a few minutes after the "fit" and then regained its feet. The hyperexcitability was still present.

After four days dosage with magnesium carbonate, clinical signs diminished and remained so throughout the continuance of dosage. After 10 days, treatment was again discontinued and clinical signs gradually returned. One gramme magnesium sulphate was then given intravenously. Twenty-four hours later, the blood serum magnesium was again below 0.7 mg.

per 100 ml. and clinical signs were still present. A single intravenous injection of 2 grammes magnesium sulphate also resulted in no apparent permanent improvement. The calf was not treated further, and the clinical signs gradually regressed throughout the next month, but the calf did not grow very well. This was probably due to the acceleration of energy metabolism which occurs in the disease (Blaxter & Rook, 1953).

Blood samples taken from the remaining calves showed that besides calf A, calf B and calf D had serum concentrations of magnesium below 0.8 mg. per 100 ml. Calf L, however, maintained a value of 1.5 during a three week period of observation, and calf K values greater than 2.1. This marked variation between calves is quite typical of a number of farms studied and makes the diagnosis of the cause of death from a consideration of the blood composition of survivors in the same herd impossible.

Clinical signs were only seen in two calves, A and B, those with the lowest serum levels. Those in calf A are described in detail. The serum magnesium levels on May 20th were: A, −0.62; B, −0.42; D, −0.54; F, −0.96; R, −0.81; L, −1.44; K, −2.57. Serum calciums varied from 10.9 to 11.8 mg. per 100 ml. The blood from the dam of calf A contained 9.0 mg. calcium and 2.5 mg. magnesium.

In the field with its dam, the calf A was constantly on the move. When standing it "paddled" from leg to leg, switched its tail, the nictitating membrane was clearly seen when it blinked, and the ears were constantly moved, not necessarily in response to sound. An unexpected sound such as clapping of the hands sometimes produced a jump. Passage of urine was normal. At the trot, fore-legs were lifted abnormally high, rather like a Hackney horse, the eyes protruded, the nostrils were dilated, and the head was held high. At the gallop the calf had the appearance of wild excitement and there was frothing at the mouth.

Close examination of the calf in this hyperexcitable stage demonstrated accelerated pulse and respiratory rates, normal temperature, exaggerated reaction of subcutaneous muscles of chest and flanks to touching the mid-line hairs of the withers, and a generalised skeletal muscle jerk on administration of a gentle tap to withers or loin. In addition clonus of the muscles of the arm and fore-arm was evoked by holding the fore-arm horizontal, allowing the cannon to swing, and giving a sharp tap to an extensor tendon as it traversed the carpus. Similarly, clonus of the thigh muscles was provoked by tapping the middle patellar tendon. Repeated blinking was stimulated by a single light blow on the facial nerve.

Calf B was observed to move its ears constantly, to have protruding eyes and to give exaggerated skin responses. Its gait was, however, normal. (Serum magnesium 0.97 mg. per 100 ml.)

Farm DC

This is a marginal farm consisting of 300 acres arable and 4,000 acres of windswept peat moorland, growing rough grass, heather and rushes. The farm has been steadily improved during the last 20 years by application of lime and phosphates to parts of the moor, by careful manuring and cultivation of the arable land and by steadily increasing the cattle and sheep in balanced proportions. In 1933 there were 600 breeding ewes and 12 breeding cows. Now there are 1,000 breeding ewes and 170 breeding cows.

In winter and spring the cattle are housed at night in cattle courts and by day, except in severe weather, they are turned out to the moorland. Straw and turnips are fed. The cows calve from December to April on the moor by day or in the courts by night. Each cow suckles only one calf.

Jamieson and Allcroft demonstrated that Copper Pine retarded the growth-rate of the calves on this farm (Jamieson and Allcroft, 1950: farm D), and since then summer dosing of the calves with copper sulphate has been carried out.

A few sudden deaths have occurred each spring, supposedly due to "hairball." This year five calves died in the first three weeks of March, all in the two to three and a half month age group. The last two of these were examined. Neither contained any foreign material in the abomasum. One had five circular haemorrhages about 1 cm. in diameter beneath the serous covering of the aorta. Otherwise no abnormalities were detected. The second, which died 36 hours before examination, did not have any haemorrhages on the great vessels. The Ca : Mg ratio in metacarpus and rib, however, was very high in each case (see cases DC 54/1, DC 54/2, Table II).

About 20 of the 50 calves remaining in this group were found to be exhibiting clinical signs of hypomagnesaemia in all degrees of intensity. Six calf bloods were taken at random on this and three other occasions. Daily oral treatment with 1 gramme magnesium carbonate was carried out from March 27th until April 17th (three weeks).

The following analytical data were obtained from calves picked at random from the group.

March 26th—0.5, 0.6, 0.7, 0.8, 1.4, 2.0 (mean 1.0).

April 5th—0.6, 0.7, 0.8, 0.9, 1.2, 1.7, 1.9 (mean 1.1).

April 27th—0.6, 0.9, 1.0, 1.3, 1.6, 2.2 (mean 1.3).

May 25th—0.7, 0.9, 1.1, 1.8, 1.9, 2.0 (mean 1.4).

Two calves were not dosed and one of these died. The treated calves, though they showed no spectacular increase in serum magnesium, suffered no losses during the period. The farmer reported that after three days of daily treatment there was a marked improvement clinically in the group. The calves became much more restful and they throve better. On each of the first two round-ups for dosing one calf collapsed in tetanic convulsions, but regained composure and survived. There were no convulsive seizures after the second day of dosing.

After two weeks' dosing, the cattle were turned into a field of first-year grass and after a further week dosing was discontinued. A week later another death occurred in this dosed group. This calf was not examined by us, but the rib and metacarpus was obtained. Analysis showed severe magnesium depletion (DC 54/108, Table II).

Clinical Signs as Observed in the Field

The signs associated with very low concentrations of magnesium in the blood serum, are identical with those described in calves in specific magnesium depletion under experimental conditions (Blaxter, Rook and MacDonald, 1954). It has proved possible with experience, to find very slight clinical signs of neuromuscular irritability in animals with a magnesium content of the serum between 0.8 and 1.0, but a characteristic clinical syndrome only develops when the serum magnesium is depressed below 0.8 mg. per 100 ml., and is most severe when the concentration has fallen to 0.6 and below. Apprehension, movement and changes in the carriage of the ears, an occasional tendency to "star-gaze," a rather stilted exaggerated movement of the limbs, and a wide palpebral fissure are the signs seen on observation of the animal in its pen. The constant movement of the ears has been found to be the most easily recognised clinical sign observed when a group of calves is scrutinised. Close examination indicates an absence of an elevated body temperature, an accelerated pulse rate, a positive reaction on tapping the facial nerve, exaggerated skin reflexes, and most diagnostic, perhaps, grossly exaggerated tendon reflexes with clonus.

Post-Mortem Appearance

In calves which have died following convulsions associated with hypomagnesaemic tetany the findings have been gross terminal haemorrhage particularly of the walls of the great vessels and of the endocardium. In none of the animals examined has any pathological calcification of the myocardium, the blood vascular system or the kidney been observed and in several no pathological changes have been noted at all. Calcification of the musculature has been found, however, in some calves dying in the area, and this aspect of the "milk syndrome" is being studied further. The results of pathological examination do not therefore agree with the description of so-called magnesium deficiency by Moore *et al.* (1938) where gross calcification was the predominant sign. They are in perfect agreement, however, with the recent experimental observations made at the Hannah Institute in which magnesium deficiency uncomplicated by other nutritional or infectious conditions was produced. (Blaxter, Rook and MacDonald, 1954a.)

The Magnesium Content of the Bones

The most reliable method of confirmation of hypomagnesaemic tetany has been found to be the analysis of the bones after death of the animal. This aspect of diagnosis is of considerable importance since often farmers fail to realise that any abnormality is present in their animals until one dies suddenly in convulsions. Bone analysis can establish whether or not magnesium tetany was the cause of death. Table II summarises analyses of bones from animals dying in the area, not necessarily of hypomagnesaemic tetany. Diagnosis of muscular dystrophy was based on clinical signs and *post-mortem* appearance, confirmed by biochemical and histological examination of the muscles.

The results for the last four animals in Table II are for experimental calves, two normal, one succumbing very rapidly to magnesium deficiency and one exhibiting chronic tetany for a long period (30 days). They indicate that values below a 60 : 1 ratio of Ca : Mg may be regarded as normal, values above 90 as indicative of severe bone depletion, the higher the elevation above 90 : 1 the greater the presumptive period of depletion. On this basis, calves NHM 53/2, AGM 53/2 and NP 54/1 are clearly normal, while all calves from farm DC were severely depleted. Calf C 144/54 may be regarded as probably a mixed condition. Blood serum from calves NP 1/54, NHM 53/2 and AGM 53/2 contained over 1.9 mg. magnesium per 100 ml. serum.

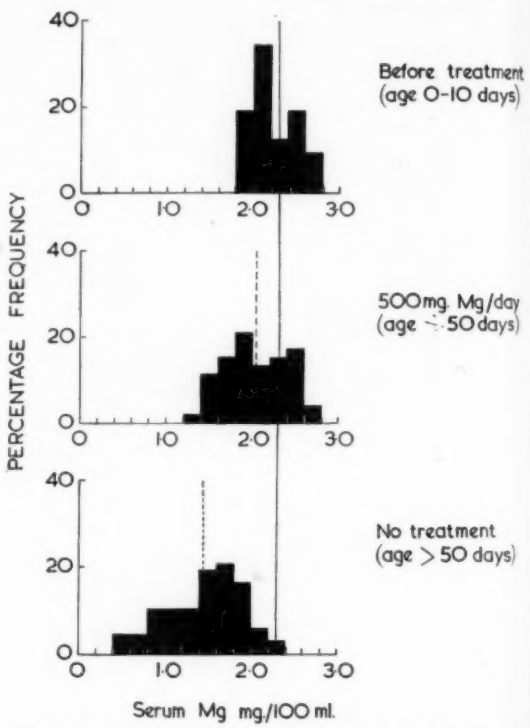

FIG. 3.—Percentage frequency distribution showing the effect of daily dosage with 500 mg. magnesium as the carbonate on the blood serum magnesium concentration of calves. Eighty calves were given magnesium and 80 were controls.

Prevention of Hypomagnesaemia

In 1954 approximately 80 calves on 20 farms were given 500 mg. magnesium each day as the carbonate and a further 80 calves on the same farms, comparable as far as possible with respect to age, received none. Dosing usually commenced during the first 10 days of life and continued until the animals went with their dams to grass in the spring. The period of dosage thus varied, from a minimum of a month in late-born calves to two and a half months in those born early in the year. In Fig. 3 the mean results are given for calves aged 50 days and more, which had received treatment for the whole period. It will be noted that the undosed calves showed the expected fall in blood serum magnesium, some 19 per cent. having a blood serum magnesium below 1.0 mg. per

100 ml., 9 per cent. being in the dangerous tetany range, and 4.5 per cent. showing recognisable severe clinical signs of the disease necessitating treatment. Of the calves which were dosed, however, only 2 per cent. fell in the range 1.2 to 1.39 mg. per 100 ml., the mean level of the group was 2.0 mg. per 100 ml. and there were understandably no signs at all of any neuro-muscular disturbance.

The dose of 500 mg. daily did not, however, ensure that the blood serum magnesium of each individual was kept at the so-called normal level of over 2.0 mg. per 100 ml. In Table III, the mean results for

TABLE III

MEAN SERUM CONCENTRATION OF MAGNESIUM OF CALVES GIVEN 500 MG. MAGNESIUM AS A SUPPLEMENT TO THEIR RATION EACH DAY FOR MORE THAN 50 DAYS

Number of farms	Total Number of calves	Mean concentration of magnesium in serum (mg. per 100 ml.)		
		Control calves	Dosed calves	Response
3	20	2·08	2·20	+0·14
3	32	1·75	2·00	+0·25
6	52	1·53	1·94	+0·41
7	52	1·24	1·73	+0·49

all calves have been classified according to the mean serum magnesium found in undosed animals on the farms concerned. On farms where control calves showed no fall in the serum magnesium, magnesium dosage nevertheless gave rise to a small increase in the magnesium concentration in the serum. Where a considerable depression of serum magnesium occurred, however, the response was considerably greater, but not sufficient to maintain the serum concentration at a level comparable to that observed on farms experiencing no depression at all.

Further examination of the data showed that the dosage of magnesium employed was quite adequate to maintain serum magnesium concentration above 2 mg. on "susceptible" farms for at least the first month and that it was only after one month that any fall in concentration occurred. Even so, the small dose of magnesium given certainly prevented any clinical signs or losses of calves, and though 2 per cent. of the dosed calves did in fact have serum levels in the range 1.2 to 1.39, this had no discernible effect on their well-being.

Some of the calves in the above experiment not included in Fig. 3, did not commence treatment until they were more than a month old. Some of these calves had low concentrations of magnesium in their serum initially and dosage with 500 mg. instituted late did not necessarily prevent a further decline. Thus one calf initially, at the age of 50 days had a blood serum magnesium concentration of 1.37 mg. per 100 ml. Following 35 days' dosage, the concentration had fallen to 1.28. This and similar results tend to confirm the observations that an additional 500 mg. of magnesium each day is not sufficient to maintain normal serum values in animals aged two months or more.

The Serum Calcium in the Disease

Fig. 4 shows that the serum calcium of the calves remained in a relatively small frequency range until the serum magnesium level had fallen to below 0.8 mg. per 100 ml. Then a proportion of the calves showed a concommitant hypocalcaemia. These are comparable to the experimental calves of Parr & Allcroft (1953). This presumably reflects complex readjustments of ionic equilibrium; certainly it does not reflect any concommitant abnormality of calcium nutrition or a generalised demineralisation of bone. Bone calcium in fact was slightly elevated. It may be noted in passing, that the serum calcium of these

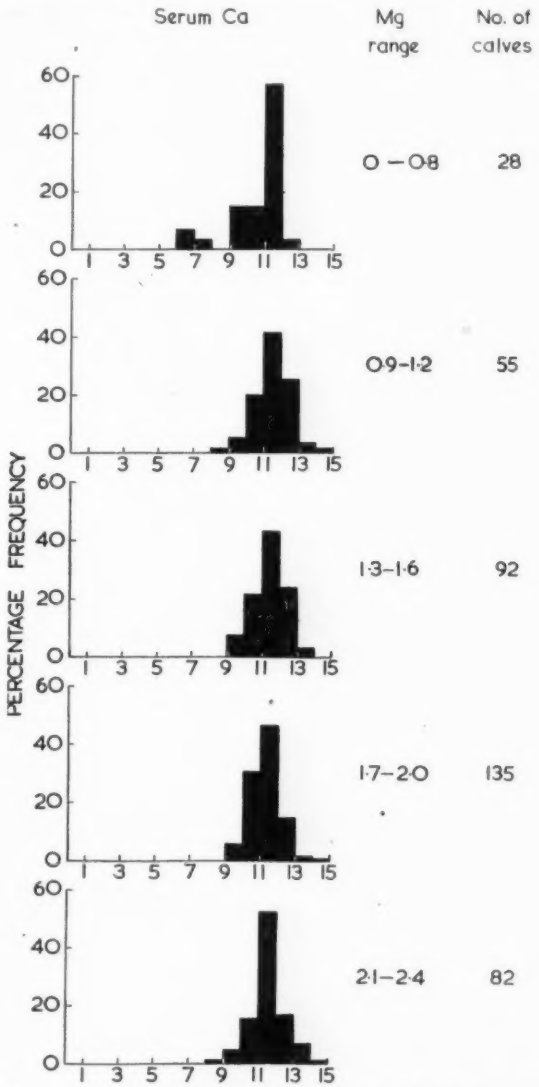

FIG. 4.—Percentage frequency distributions showing the serum calcium concentration of calves with differing levels of serum magnesium. Note a slight tendency for the calcium to be depressed when the serum magnesium is very low.

calves receiving liberal amounts of milk was much higher than the values of 10 to 11 mg. per 100 ml. obtained in cows.

Discussion

The present results leave little doubt that on the beef-rearing farms of Inverness hypomagnesaemia in calves is extremely common. The neuro-muscular irritability to which it gives rise is a condition quite distinct from muscular dystrophy, clinically, pathologically and biochemically. The two diseases occur with an overall incidence of 25 per cent. and approximately 5 per cent. respectively on the farms as a whole, though on individual farms, very heavy losses may be occasioned by one disease while the other is absent. Thus Farm DC had never experienced any trouble with muscular dystrophy yet lost many calves as a result of tetany; Farm NP, however, lost calves as a result of muscular dystrophy only.

The disease causing tetanic convulsions and associated with a lowered concentration of magnesium in the serum has been shown to be exactly comparable to the disease produced in the laboratory by feeding magnesium deficient rations to calves (Blaxter, Rook & MacDonald, 1954a). There is ample evidence that the disease in the Inverness calves is also of nutritional origin and is indeed a deficiency disease using this term to indicate a dietary inadequacy. There is no need to postulate any endocrine, neurological or other hypothetical and ill-defined physiological upset to explain its occurrence.

Unpublished observations (Sharman, Hutcheson & Blaxter, 1954) have shown that there is a positive association between the amount of magnesium in the milk ingested by individual calves and their blood serum magnesium concentration, and that calves which grow the fastest as a result of a high calorific intake from their dam's milk usually show the greatest depression of serum magnesium. This latter effect has been noted also on Farm DC, where the calves from the best milking cows died or developed tetany and calves of the poor milking beasts did not. The rate of gain of these calves is great. Commonly gains are well over 1½ lb. daily for long periods, and muscular development is considerable; milk intakes of well over 20 lb. per day have been recorded (Sharman, Hutcheson & Blaxter, 1954).

It is possible to make an estimate of the magnesium requirements of these calves. The gain of 1½ lb. bodyweight daily would entail the daily storage of approximately 450 to 500 mg. magnesium, probably more since the ratio of muscle to bone development is so high. The minimal net requirement for maintenance would be 3 mg. per kg. bodyweight, judged from the metabolic balances of Blaxter and Rook (1954). This would give a tentative net estimate of requirement of 700 mg. for a young calf aged one to two months. The balance experiments quoted above indicate that the availability of the magnesium of milk at the maintenance level of nutrition is 65 per cent. (mean value). When the calf is storing magnesium the retention is considerably less efficient, dropping in fact to lower than 30 per cent. as the animal's daily retention increases to the maximal retention attainable. The net requirement of 700 mg. would therefore necessitate a dietary intake of 1,400 mg. per day, probably more. Many of the calves do not receive half this amount in the 20 lb. or so of milk they consume each day.

Further evidence that the condition is indeed a deficiency disease is given by its prevention by a dietary supplement of 500 mg. of magnesium per day. This was sufficient in most instances to maintain normal serum concentrations and certainly to prevent clinical signs. Finally, the response of depleted calves to a single injection of magnesium is transitory—unlike the milk fever condition in the cow where a single injection apparently restores a disturbed equilibrium. This is understandable, for the following reasons. One injection of 1 gramme magnesium sulphate ($MgSO_4 . 7H_2O$) in a calf receiving a deficient ration would supply 99 mg. magnesium, that is only 14 per cent. of its tissue requirements for a single day and would have no effect whatever in restoring the skeletal magnesium, which may show a deficit of 20 grammes, to a normal value. Even the usual veterinary dose of ¼ oz. would only supply the daily requirement. Naturally, a clinical response would take place since the extra-cellular concentration of magnesium is raised (Blaxter, Rook & MacDonald, 1954b). This, however, would not be maintained and a relapse would occur. That this sequence does in fact take place is evident from the results obtained with calf A on Farm L. It is legitimate to speculate that the numerous breakdowns observed in cows with lactation tetany given intravenous magnesium is also due to failure to restore the depleted exchangeable magnesium of bone. Certainly, magnesium metabolism experiments at the Hannah Institute with milking cows may be interpreted in this way (Blaxter, Presthegge & Wood, 1950).

Similarly, daily dosage with 500 mg. magnesium (about one third of the daily requirement) would not restore a depleted skeleton if the animal's diet remains deficient. This is shown by the behaviour of calves with initially low serum magnesium concentrations in the field experiment, and, more forcibly on Farm DC. Here daily dosage for three weeks stopped all losses but one week later a calf died of the disease and its bones were still depleted of the element. Whether very much larger amounts of magnesium than we have given can be tolerated for long periods without producing digestive signs or systemic disturbances remains to be seen. It is quite probable that they can, but care is clearly necessary.

Summary

(1) Studies on 25 farms in Inverness and one in Caithness in 1953 and 1954 show that a slow fall in the concentration of magnesium in the blood serum of suckling calves takes place during the first three months of life. Some farms are affected to a greater extent than others, and on a limited number, tetany and death occur.

(2) The clinical condition is shown to be quite distinct from enzootic muscular dystrophy which is rife in the area, and its incidency is tentatively placed at 5 per cent. of all farms.

(3) Clinical signs are described, *post-mortem* appearance is briefly discussed, and the value of bone analysis in ascertaining the cause of sudden death is shown. Two farms experiencing severe "outbreaks" are described in detail.

(4) Experiments with 160 calves are briefly described which show that daily dosage with 500 mg. magnesium as the carbonate will prevent a very large part of the fall in the serum concentration of magnesium and abolish the incidence of tetany.

(5) The disease does not appear to be associated with abnormalities of calcium metabolism, and the response to a single injection of magnesium is not spectacular.

(6) It is shown that the disease is due to a simple dietary deficiency engendered by very high rates of growth on whole milk which is not a sufficient source of magnesium. Estimates of requirements are made to illustrate this point.

(7) Small amounts of magnesium given by vein or by mouth have only a temporary effect in abolishing tetany. The quantities given, while not small in terms of daily requirements are small in terms of the overall body deficit of magnesium.

Acknowledgments—It is a pleasure to record our thanks and appreciation to the co-operating farmers in the district who so willingly have helped us in this work, to Miss M. K. Hutcheson for her help with the field work in 1954, and to Miss Grace Breckenridge and Miss Maimie Jack for their technical help at the laboratory. We are grateful to Messrs. Roche Products for supplying the magnesium tablets.

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ADVERTISER'S ANNOUNCEMENT

Glaxo Laboratories Ltd. announce the introduction, from January 24th, of a half-strength Streptopen Uterine Tablet which will be known as "Streptopen '250' Uterine Tablets." These tablets are primarily intended for *post-partum* infections in ewes and sows.

The existing pack of Streptopen Uterine Tablets will in future be known as "Streptopen '500' Uterine Tablets."

HYPOTHERMIA

In human surgery, increasing use is being made of hypothermia for operations within the dry heart, and the techniques have been based upon experimental work carried out upon dogs. Research workers found that if the dog's body temperature was reduced to 68° F., the inflow of blood could be interrupted long enough for the heart to be opened and examined. In an early series of experiments it was found possible to close atrial septal defects under these conditions, and the technique was used successfully on a five-year-old girl suffering from such a lesion. In her case, after being anaesthetised, she was wrapped in refrigerated blankets for two hours and 10 minutes until the body temperature had dropped to 82.4° F. The inflow of blood to the heart was stopped for five and a half minutes and repair of the septal defect was carried out under direct vision. The operation lasted just under one hour, and at the end of it she was immersed in hot water to restore the body temperature to normal.

The use of refrigerated blankets or of ice packs has certain disadvantages, and in order to overcome these a new method of cooling the bloodstream was evolved. After applying in 130 dogs an existing technique of inducing hypothermia by cooling the arterial blood in a circuit outside the body, a similar method of cooling the venous blood was resorted to. The apparatus consists of a coil of Polyvinyl tubing, about 12 feet long, surrounded by a circulating refrigerant solution. A simple rotary pump drives the venous blood through the cooling coil and back into the body. The dog's external jugular vein is exposed through a small skin incision in the neck, and a cannula gently manipulated into the superior vena cava. The cooled blood is returned via a similar catheter inserted into the femoral vein through the saphenous vein. When the pump is started, cooling begins, and it takes about 25 minutes to cool a 44 lb. dog to 77° F. As soon as this is reached, the cannulae are removed, leaving a clear field for the operation. This technique has since been successfully applied in the case of humans.

Hypothermia has, almost certainly, no application in veterinary surgery, and only a limited one—not free from several attendant risks—in human surgery, but the fact that preliminary work was carried out upon dogs will be of interest to the veterinary profession.

Public Relations

At this time when a sub-committee jointly from the Royal College of Veterinary Surgeons and our own Association is discussing a number of matters with an animal welfare society, it would not be opportune to dwell unduly upon the subject of clinics and publicity; but if members of the profession would help in the ways indicated herein, a start at least could be made along the road which many veterinarians would wish to see followed, so long as it was followed ethically.

The Treatment of Chronic Carriers of *Salmonella pullorum* with Furazolidone

A PRELIMINARY REPORT BY
R. F. GORDON and J. TUCKER
Animal Health Trust Poultry Research Station, Houghton

A SEARCH of the literature indicates that there have been very few investigations into the value of drug therapy in the reduction of blood titres or in the elimination of foci of infection in carrier fowls infected with *Salmonella pullorum*. Cole (1948), however, showed that the prolonged feeding of sulphonamides (sulphamerazine, sulphadiazine and sulphaquinoxaline) did not prove effective in removing *S. pullorum* from yearling hens and survivors were found to have specific agglutinins in their blood for 180 days subsequent to the termination of treatment.

In view of recent work (Williams Smith, 1954a & b) establishing the successful use of furazolidone in the treatment of carriers of *S. gallinarum* and of chicks infected with *S. pullorum*, the effect of this compound on naturally occurring carriers of *S. pullorum* was considered worthy of investigation.

A total of 22 adult birds which had given positive results to the rapid whole blood agglutination test in the field were obtained from a number of farms and divided into two groups. Group I consisted of seven fowls, four of which were treated for 10 days by the addition of furazolidone in the food at a level of 0.02 per cent., while the remaining three birds were retained as untreated controls. Group II contained 15 birds, nine of which were fed furazolidone continuously in the mash for 10 days at a level of 0.04 per cent.; three birds (one from each of the three farms involved) were sacrificed before treatment started, in order to confirm the presence of *S. pullorum* infection, while three birds were again retained as untreated controls. The bacteriological examination of cloacal swabs and of the carcases on autopsy was by the method described by Williams Smith (1954b). Agglutination tests by the serum tube method were carried out before and immediately after treatment and then at weekly intervals until the termination of the experiment. Cloacal swabs were taken daily during the treatment period and thereafter twice weekly. The results are shown in Tables I and II.

Results

It will be seen from Table I, that three of the four treated birds showed no evidence of *S. pullorum* or other *Salmonella* infection on autopsy, while two birds showed a marked reduction in serum titre. One of the treated birds and all the controls maintained their serum titres and yielded *S. pullorum* from a variety of sites, including the reproductive organs. All 91 cloacal swabs taken from this group were negative for *Salmonella* on bacteriological examination.

TABLE I
BIRDS FED 0·02 PER CENT. FURAZOLIDONE FOR 10 DAYS

Bird number	Serum titre			Time lapse between end of treatment and killing (in days)	*Post-mortem* findings	Bacteriological examination
	Before treatment	After treatment	When killed			
1	1 : 250		1 : 250	Untreated control, killed 43 days after arrival at laboratory	Degenerated ovary	*S. pullorum* from heart, spleen, liver and gall bladder
2	1 : 250		1 : 250	Untreated control, killed 93 days after arrival at laboratory	Degenerated ovary, malformed egg in oviduct	*S. pullorum* from spleen, ovary, oviduct, intestines and malformed egg material
3	1 : 25		1 : 25	Untreated control, killed 43 days after arrival at laboratory	Tumour in oviduct, otherwise normal	*S. pullorum* from oviduct and intestines
4	1 : 250	1 : 250	1 : 250	83	Degenerated ovary	*S. pullorum* from spleen, ovary and oviduct
5	1 : 250	1 : 250	1 : 25	83	Degenerated ovary	Negative
6	1 : 250	1 : 250	1 : 250	34	Degenerated ovary	Negative
7	1 : 50	1 : 25	1 : 10	33	Normal	Negative

Table II shows that seven of the nine treated birds became negative to the serum tube agglutination test between 60 to 84 days after the cessation of treatment. All these birds originally had titres of at least 1 in 125. Five of the fowls which became negative to the agglutination test had had titres of at least 1 in 250, the highest titre found. In one further bird, destroyed two days after the end of the treatment, the titre had also shown a significant drop from 1 in 250 to 1 in 50. This rapid initial drop in serum titre soon after the cessation of treatment was observed in the majority of treated birds. No decrease in titre occurred in any of the untreated controls.

Eight of the nine treated birds were found to be free from *S. pullorum* infection on autopsy, including the bird killed two days after treatment terminated. The bird which failed to respond to treatment and all six untreated controls yielded *S. pullorum* from the reproductive organs and other sites.

Of 91 cloacal swabs from the control birds, two, both from the same bird (No. 13), yielded *S. pullorum*. *S. pullorum* was recovered from one out of 254 cloacal swabs taken from the treated group. This positive swab came from bird No. 18, which failed to respond to the drug. Since this bird retained its serum titre, in practice it would have been removed from the flock by routine blood testing.

It is important to note that the majority of the successfully treated birds in both groups showed degeneration of the ovary, characteristic of *pullorum* disease and indicative of past *S. pullorum* infection.

Conclusions

When furazolidone at both 0.02 per cent. and 0.04 per cent. was fed continuously for 10 days in the rations of 13 birds positive to the rapid whole blood test in the field, 11 were subsequently found to be free from *S. pullorum* infection. All of the

TABLE II

BIRDS FED 0·04 PER CENT. FURAZOLIDONE FOR 10 DAYS

Bird number	Blood titre			Time lapse between end of treatment and killing (in days)	*Post-mortem* findings	Bacteriological examination
	Before treatment	After treatment	When killed			
8	1 : 250		1 : 250	Untreated control, killed on arrival at laboratory	Normal	*S. pullorum* from liver and ovary
9	1 : 250		1 : 250	Untreated control, killed 94 days after arrival at laboratory	Degenerated ovary	*S. pullorum* from heart, spleen, gall bladder, liver, ovary and oviduct
10	1 : 125		1 : 125	Untreated control, killed on arrival at laboratory	Normal	*S. pullorum* from spleen, gall bladder, liver, ovary oviduct and intestines
11	1 : 250		1 : 250	Untreated control, killed 52 days after arrival at laboratory	,,	*S. pullorum* from heart, ovary and intestines
12	1 : 250		1 : 250	Untreated control, killed on arrival at laboratory	Cystic ovary	*S. pullorum* from heart, spleen, liver, ovary, oviduct and intestines
13	1 : 250		1 : 250	Untreated control, killed 92 days after arrival at laboratory	Degenerated ovary	*S. pullorum* from ovary and oviduct
14	1 : 250	1 : 50	1 : 50	2	Normal	Negative
15	1 : 250	1 : 50	-ve 1 : 10	70	Degenerated ovary	,,
16	1 : 250	1 : 50	-ve 1 : 10	84	Normal	,,
17	1 : 250	1 : 125	-ve 1 : 10	70	Degenerated ovary	,,
18	1 : 125	1 : 50	1 : 250	84	Degenerated ovary	*S. pullorum* from heart, spleen, gall bladder, liver, ovary and intestines
19	1 : 250	1 : 250	-ve 1 : 10	60	Degenerated ovary	Negative
20	1 : 125	1 : 125	-ve 1 : 10	70	Normal	,,
21	1 : 125	1 : 125	-ve 1 : 10	70	Degenerated ovary	,,
22	1 : 250	1 : 250	-ve 1 : 10	60	Normal	,,

untreated controls showed generalised *S. pullorum* infection. When the drug was used at 0.04 per cent. seven out of eight birds became negative to the agglutination test 60 to 84 days after the termination of treatment. Weekly testing indicated that there was an initial decrease in serum titre immediately treatment had been completed, and one bird examined two days after the end of treatment failed to yield *S. pullorum*.

Further laboratory investigations are in progress, using considerably larger groups of *S. pullorum* carriers, together with extensive field trials as to the value of the drug as a supplement to the rapid blood test in eliminating carriers from infected hatchery supply flocks.

In the results to date the drug has shown very considerable promise in this respect. When treatment has been applied to heavily infected flocks after routine blood testing and removal of reactors, subsequent testing at one month's interval has revealed few, and in the majority of treated flocks, no reactors. It should be stressed that in these field trials the drug was used at a level of 0.04 per cent. and all food, other than the medicated mash (*e.g.* grain, etc.), was witheld during the period of treatment. Previous experience has shown that in some heavily infected flocks testing and retesting can be prolonged and that fowls recently infected or with a low serum titre may escape detection. The results reported would strongly suggest that once positive reactors are removed by routine testing, treatment of the remainder of the flock with furazolidone should be an added insurance of freedom from infection in hatchery supply flocks.

The full implication of the use of the drug in this respect and in the treatment of reactors retained for commercial egg production will be discussed when the final results of present experiments and field trials are available at the end of the present breeding season.

Acknowledgments—We wish to thank the various hatcheries who have arranged for the field trials and for the supply of *pullorum* carriers. We also thank Messrs. A. J. White, Ltd. for the supply of furazolidone and Miss Jill Armstrong for technical assistance.

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MR. W. PEIRSON

We are indebted to a correspondent in New Zealand for a note on the retirement of Mr. W. Peirson, a distinguished member of the profession in that country. Mr. Peirson has been in practice for 30 years, 28 of them in New Zealand, and at a function in his honour, Mr. J. A. Roy, M.P., paid tribute to his work for the farming community. A number of other speeches were made by veterinarians, public officials, and agriculturalists, and a presentation to Mr. Peirson was made.

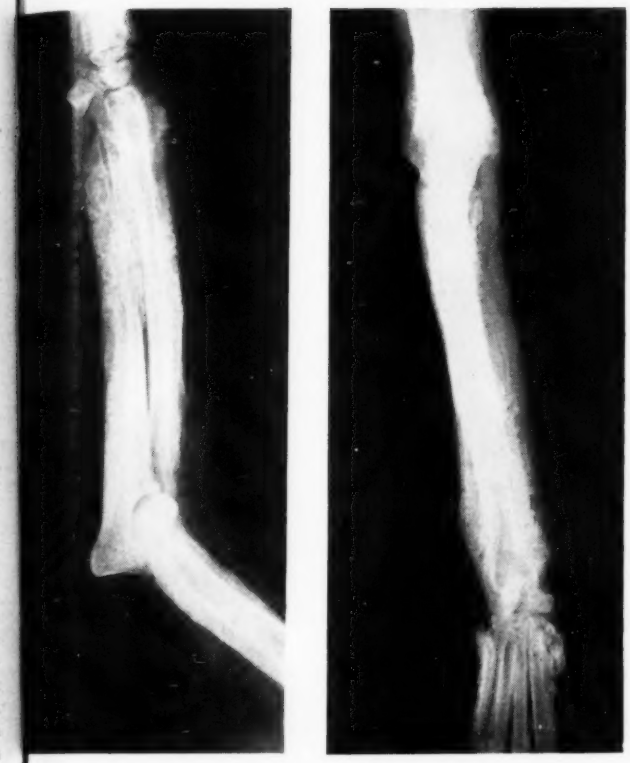
Hypertrophic Pulmonary Osteoarthropathy in the Dog associated with Tuberculosis

BY
A. H. HOGG
Leamington Spa
AND
H. HOLROYD
Blackburn

Introduction

THIS condition is rare in the dog but several cases have been reported at intervals (Cotchin, 1944; Craig & Davies, 1940; Innes, 1940; Wooldridge & Holmes, 1937; Smythe, 1929). It has also been described in Continental literature and by Schnelle and Poley & Taylor in America. It is variously described as Marie's disease, acropachia, hypertrophic periostitis, etc. In man the bone lesions may be secondary to various chronic lung conditions but in the dog the majority of the cases described have followed tuberculosis. The cases described by Schnelle and Poley & Taylor, however, followed tumour of the lung. In a recent American case the lung lesion was identified as being a fibrosarcoma. American references to this

FIG. 1.—Marked thickening of heck and tarsal bones.



HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY IN THE DOG ASSOCIATED WITH TUBERCULOSIS

PLATE I

PLATE II

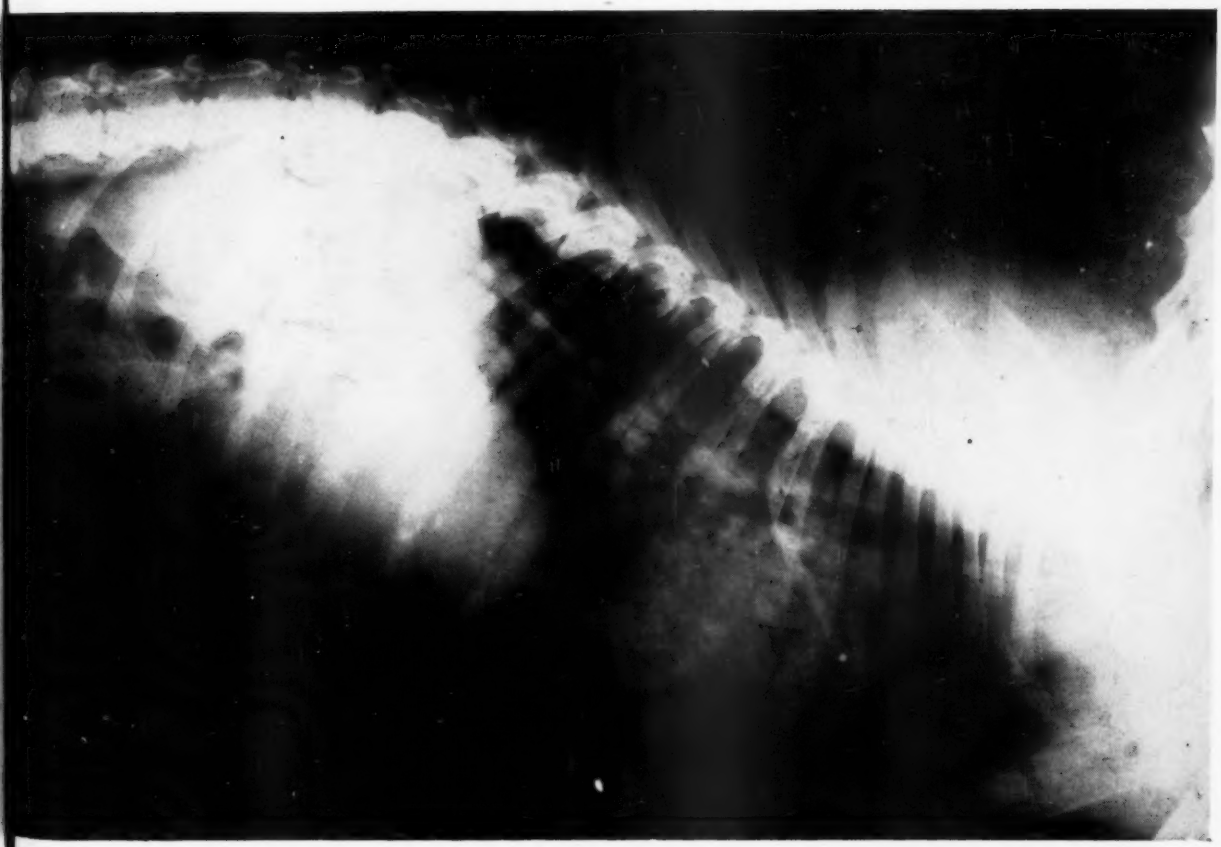


FIG. 2.—Thickened radius, ulna and carpal bones.

complaint associate the bone lesions with a lung neoplasm. In this country the lung condition has generally been that of tuberculosis.

Our main justification for publishing the details of yet another case which differs but slightly from those previously described in this country at least (England), is that more is known in this instance regarding certain aspects of the case through the fact that the dog was hospitalised for a period of six weeks prior to death.

Subject.—Two-year-old farm collie. Male.

History.—One of us (A. H. H.) observed the dog whilst visiting a farm in connection with large animal work. Comment was passed regarding the abnormal thickness of the legs and joints (Figs. 1 and 2). This elicited the opinion from the owner that the dog had been suffering from distemper and that the thick legs were a direct consequence of that disease. This explanation was not unreasonable for in fact at that stage, apart from its limbs, the dog resembled closely a case of advanced distemper with marked respiratory complications. Permission was obtained to radiograph the limbs, and a diagnosis of pulmonary osteoarthropathy followed immediately (Plate I). Since the only point remaining was of academic rather than practical importance, namely, the exact diagnosis of the chest lesion, the owner was asked what he would like done. His reply being non-committal it was decided to board the dog with a view to further radiography, this time of the chest. The results suggested pulmonary tuberculosis (Plate II) and the intradermal tuberculin test which was subsequently performed showed a marked and obvious positive reaction by the 48th hour. It had been suggested that a trial might be made with Isoniazid but a week having elapsed since the dog was taken into hospital it became apparent that a natural improvement was taking place in the animal's general condition. The drug was therefore never used. The apparent improvement was maintained for over a month when a rather sudden and marked worsening of the chest condition necessitated destruction on humane grounds.

Post-mortem Findings.—The autopsy revealed extensive tubercular disease affecting both lungs with cavitation a prominent feature. A few small lesions were found in the mediastinal lymph glands but in no other organs. Smears from the lung revealed typical acid-fast bacilli.

The owner had informed us, before a diagnosis was made, that the dog had been generously fed with milk from puppyhood. One of us (H. H.) had discovered and slaughtered on this particular farm a cow affected with udder tuberculosis approximately one year before the dog's illness. According to the owner the first symptoms of this illness were shown about six weeks before we saw the dog. A medical radiologist diagnosed the chest condition without previous prompting, saying that the fuzziness or woolly appearance was typical of advanced consumption in the human. Another noteworthy feature was the presence of cavitation in the lung. This is also a feature of the human condition but unusual to veterinary practitioners accustomed to the appearance of the disease practically only in the bovine, in which species, of course, cavitation occurs rarely, if at all.

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PARIS ART EXHIBITION

The 27th Art Exhibition for doctors, dentists, pharmacists, and veterinary surgeons will be held between March 6th and 20th, at the Musé d'Art Moderne, Paris. The Minister of Public Health will open the exhibition, and as in past years will award medals to those who sent the best paintings, sculptures, decorative art, photography, and applied art. Exhibitors should communicate details, name, qualifications, address, and title of object to the secretary: Doctor Malet, 67, Avenue Pierre Larousse, Malakoff (Seine), France. The same details should appear on a label attached to the object and sent to: Messrs Davies Turner Ltd., 4, Lower Belgrave Street, London, W.1, and *not* direct to Paris. The secretary of the exhibition extends a cordial welcome to British veterinarians.

News and Comment

AGRICULTURAL RESEARCH COUNCIL

The Agricultural Research Council, in agreement with the University of Cambridge, has established at Cambridge a Unit of Reproductive Physiology and Biochemistry. Dr. T. R. R. Mann, M.D., SC.D., PH.D., F.R.S., Reader in the Department of Veterinary Clinical Studies, has been appointed Director of the Unit. There will be two Deputy Directors—Dr. A. Walton, B.SC., PH.D., and Mr. L. E. A. Rowson, M.R.C.V.S. Mr. Rowson is also Director of the Artificial Insemination Centre of the Cambridge and District Cattle Breeders Ltd.; his services to the Unit will, therefore, be part-time.

FOWL PEST IN NORFOLK

In view of the occurrence recently of numerous outbreaks of fowl pest in the Great Yarmouth area of Norfolk it has been decided to extend the present scheduled area. The new area is as follows:—

The City and County Borough of Norwich.

*The County Borough of Great Yarmouth.

In The County of Norfolk—The petty sessional divisions of Eynsford, Mitford and Launditch, *South Erpingham, *Tunstead and Happing, *Taverham, *Blofield and Walsham, and *East and West Flegg.

Petty Sessional Division of Forehoe—The parishes of Honingham, Easton, Marlingford, Barnham Broom, Runhall, Barford, Kimberley, Wramplingham, Wicklewood, Deopham, Morley, and Hingham.

Petty Sessional Division of Guiltcross and Shropham—The parishes of Great Ellingham and Rocklands.

Petty Sessional Division of Wayland—The parishes of Carbrooke, Scoulton, Watton, Griston, Caston, Thompson, Little Ellingham, Merton, and Stow Bedon.

County of East Suffolk. Petty Sessional Division of Mutford and Lothingland—The parishes of *Burgh Castle and *Bradwell.

*New areas.

R.C.V.S. LIBRARY

Will readers please note that the Library telephone number is **CHAncery 5822** and not as is incorrectly shown on page 69 of the Register of Veterinary Surgeons for 1954.

PERSONAL

Births

BOWEN.—On January 22nd, 1955, to Joan, wife of P. D. G. Bowen, M.R.C.V.S., Stow-on-the-Wold, Glos., a son, James.

DICKSON.—On January 24th, 1955, at Carlisle, to Nancy, wife of Ian Dickson, M.R.C.V.S., a daughter, Ann Margaret.

FARRELL.—On January 24th, 1955, at Ashbank, Race Road, Bathgate, to Fay, wife of Adam V. Farrell, M.R.C.V.S., a son; both well.

GARRETT.—On January 26th, 1955, to Gillian Ruddick (*née* Stobbart), wife of Thomas Ralph Garrett, B.SC., M.R.C.V.S., a son, Thomas Jeremy.

ORMISTON.—On January 24th, 1955, at Abingdon, Berks., to Helen (*née* Fleming, M.R.C.V.S.), wife of T. T. Ormiston, M.R.C.V.S., a son.

Marriages

MCCULLY—EAGLESON.—On January 1st, 1955, at Kildress Parish Church, William Hayes McCully, M.R.C.V.S., only son of Mr. & Mrs. S. McCully, Churchtown, Cookstown, to Gretta, youngest daughter of Mr. & Mrs. R. D. Eagleson, Orritor House, Cookstown.

WATSON—LONSDALE.—On January 29th, 1955, at Oakham, Richard Travers Watson, Highfield, Oakham, Rutland, to Tuppina, Countess of Lonsdale, of Home Farm, Market Overton, Rutland, daughter of the late Capt. G. H. Bennett & Mrs. V. B. Bennett, of Barrowby, Grantham.

R.C.V.S. OBITUARY

LAIRD.—James, of Bothwell Cottage, McArthur Street, Dunoon, Argyllshire. Graduated Glasgow, May 25th, 1897. Died December 30th, 1954.

COMING EVENTS

February

10th (Thurs.). Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 8.30 p.m.

Meeting of the Technical Development Committee, B.V.A., at 7, Mansfield Street, Portland Place, W.1, 11 p.m.

11th. (Fri.). Meeting of the Technical Development Committee, 10.30 a.m.

11th (Fri.). General Meeting of the South-east Midlands Division, B.V.A., at Franklin's Garden Hotel, Northampton, 7.30 p.m.

15th (Tues.). Annual General Meeting of the Eastern Counties Division, B.V.A., at the Bell Hotel, Norwich, 10.45 a.m.

16th (Wed.). Meeting of the Section of Comparative Medicine, Royal Society of Medicine, at the Zoological Society of London, Regents Park, N.W.1, 5 p.m.

17th (Thurs.). Annual General Meeting of the South Eastern Division, B.V.A., at the Royal Star Hotel, Maidstone, 2.30 p.m.

23rd (Wed.). Joint Meeting of the Society of Practising Veterinary Surgeons and the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 2.30 p.m.

Annual General Meeting of the Scottish Metropolitan Division, B.V.A., at the Royal (Dick) School of Veterinary Studies, 2.30 p.m.

24th (Thurs.). Meeting of the Essex Division, B.V.A., at the Essex Institute of Agriculture, Writtle, 7.30 p.m.

25th (Fri.). Liverpool University Veterinary Society Annual Ball, in the Students' Union, Brownlow Hill, Liverpool 7, 8 p.m.

Annual General Meeting of the Midland Counties Division, B.V.A., at the Birmingham Medical Institute. Business Meeting at 11 a.m., Paper commences at 2.30 p.m.

May

6th (Fri.). Royal Veterinary College 163rd Annual Ball at the Hyde Park Hotel.

September

4th to 10th. 73rd Annual General Meeting and Congress of the British Veterinary Association at Belfast.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Cumberland. Row Head, Sowerby Row, Carlisle (Jan. 24); Wallthwaite Farm, Troutbeck, Penrith (Jan. 26).

Devon. Bovingdon Hill, Plympton, Plymouth.

Durham. Long Acre Farm, Birtley (Jan. 26).

Hants. Calcot Farm, Calcot Lane, Curdridge, Southampton (Jan. 25).

Hunts. The Old Rectory, Woodwalton (Jan. 25).

Kent. Parsonage Farm, Denstroude, Blean, Canterbury (Jan. 26).

Norfolk. Church Farm, Wolferton, King's Lynn (Jan. 26).

Northumberland. Loaning Side Farm and Sunnyside Farm, Catton, Allendale, Hexham (Jan. 24); Pasture House, Catton, Hexham (Jan. 25); Houghton North Farm, Heddon-on-the-Wall, Newcastle-upon-Tyne (Jan. 26); Thistleton Farm, Thropton, Morpeth (Jan. 29).

Notts. Church Farm, North Wheatley, Retford (Jan. 29).

Salop. Top Farm, Bearstone, Market Drayton (Jan. 25).

Surrey. Rainscombe Farm, Smallfield, Horley (Jan. 25).

Warwicks. Prospect House, Prospect Lane, Solihull, Birmingham (Jan. 29).

Westmorland. Midtown Farm, Melkinthorpe (Jan. 25).

Wigtown. Dourie Farm, Port William, Newton Stewart (Jan. 26).

Yorks. Cleasby Hall, Cleasby, Darlington (Jan. 24); Carr House, Ampleforth; Rokeby Close, Barningham, Richmond (Jan. 25); Fair View Farm, Meltonby, Pocklington; Low Springs Farm, Baildon, Shipley (Jan. 27); Airedale Beagle Kennels, Spring Lane, Eldwick, Bingley (Jan. 28).

Foot-and-Mouth Disease

Northumberland. West Farm, Seghill, Dudley (Jan. 24); Lowsteads Farm, Backworth (Jan. 25); Allotment, The Old Railway, Seaton Sluice, Whitley Bay (Jan. 26); West Field, Backworth, Shiremoor (Jan. 31).

Fowl Pest

Derby. Bryntor, Moor Acre Lane, Bolsover, Chesterfield (Jan. 27).

Essex. Faggs Farm, Paslow Common, Blackmore, Ingatestone (Jan. 26); The Bungalow, Parsonage Lane, Barnston, Dunmow (Jan. 29); Springfield Hall, Springfield, Chelmsford (Jan. 30).

Herts. The Grove, Harpenden (Jan. 24); 17, Pipers Avenue, Harpenden (Jan. 27).

Hunts. 13, Green Leys, St. Ives (Jan. 30).

Lancs. Willow Farm, Greenhalgh, Kirkham, Preston (Jan. 25).

Lincs. Kirkby Underwood, Bourne (Jan. 26); Chapel Street and Station Road, Billingborough (Jan. 29).

Norfolk. Staithe House, Rollesby; Bullocks Lok, Caister-on-Sea; 10, Collis Lane, East Somerton; Pear Tree Villa, Ormesby St. Michael; 9, Johnson Street, Ludham, Gt. Yarmouth (Jan. 25); Holland Hall, Yaxham, East Dereham (Jan. 28); Prospect House, Ormesby St. Michael; 2, Wrights Buildings, Caister-on-Sea, Great Yarmouth (Jan. 30).

Surrey. Rydes Hill Lodge, Guildford (Jan. 27); Firbeck, Frog Grove Lane, Wood Street, Guildford; Lascombe, Puttenham, Guildford; Stedmans Farm, Wood Street, Guildford (Jan. 28).

Swine Fever

Bucks. Stonewell Farm, The Lagger, Chalfont St. Giles (Jan. 25).

Essex. Belle View, Peartree Green, Doddinghurst, Brentwood (Jan. 27); White Gates, Maldon Road, Birch, Colchester (Jan. 28).

Herts. Foxley Hotel, Foxley Drive, Bishops Stortford (Jan. 26); Edgwarebury House Farm, Elstree, Boreham Wood (Jan. 31).

Kent. Wilsey Farm, Cranbrook (Jan. 28).

Lancs. Suttons Farm, Drummerdale Lane, Scarisbrick, Ormskirk (Jan. 25); Bonds Hall Farm, Elswick, Preston; Maddox Farm, Wigan Road, Winstanley, Wigan (Jan. 26).

Monmouth. The Oaks, Shirenewton, Chepstow (Jan. 27).

Montgomery. Mingwern, Melfod (Jan. 25).

Northants. Hall Farm, Kilsby (Jan. 26).

Oxford. Lodge Farm, Mixbury (Jan. 28); Mill Farm, Black Bourton (Jan. 31).

Salop. Gatacre Farm, Claverley, Wolverhampton (Jan. 26); Childs Ercall Hall, Childs Ercall, Market Drayton (Jan. 31).

Staffs. High Street Farm, Knypersley Road, Norton, Stoke-on-Trent (Jan. 29).

Suffolk. Bovills Hall Farm, Gazeley, Newmarket (Jan. 27).

Worcs. Ipsley Mill Farm, Ipsley, Redditch (Jan. 25); Bittell Farm, Barnt Green, Birmingham (Jan. 28).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

A LEGITIMATE GRIEVANCE

Sir,—I have read with great interest the correspondence on this subject, and it occurs to me that the view of a layman may not be without value.

There is one aspect of the matter which I have not yet seen mentioned, and which is of the greatest importance; it is this: the fact that a person *can* pay for treatment does not mean that he *will*. There are, unhappily, many who will take their animals to a free clinic, if there is one near enough, but who will allow them to suffer if there is not, rather than pay. I am not defending these people; I merely state that they exist. By treating such cases, animal welfare societies are not really depriving practitioners of patients, and they are saving animals from suffering, which is what they are for.

In my own area I know of several animals at this moment in need of treatment which they are not receiving. Did a travelling dispensary come round here, they might get it. As it is, they will continue to pine, and scratch at their canker-stinking ears.

Further, however generous a veterinary surgeon is prepared to be to the genuinely poor, it is a hard thing to ask him for free advice. If the "almoning" at a clinic leaves much to be desired, how is the veterinary surgeon to do better? Many people these days are strained to the limit to keep up the "appearances" which may—and are intended to—delude. Even a television set may represent sacrifices for social prestige which will not be made for an animal; and cars are often provided by employers for those who certainly could not afford otherwise to run one.

The whole point is that this is not a clear-cut issue, or capable of any simple solution: it is a matter for careful and understanding discussion between both sides; and this letter is written as a contribution to that discussion.

Yours faithfully,

PETER D. TURNER,

Cave Cottage,
Oakridge Lynch,
Stroud, Glos.

January 13th, 1955.

ELECTROCUTION OF DOGS

Sir,—I hardly think it becomes Major Hume to imply that the R.S.P.C.A. asked busy scientists to answer "Yes" or "No" to problems which required scientific investigation. It is easy to be wise after the event, but, until the adoption of electrical shock methods of treatment in human beings, there was no valid reason to assume that the electrocution of dogs was not a humane method of destruction.

I think I am correct in stating that the R.S.P.C.A. initiated scientific investigation into the electrical stunning of pigs with the aid of eminent scientists and, I believe, in order to do so it must have been obligatory for the R.S.P.C.A. to obtain the necessary licence for such experimentation. The R.S.P.C.A. was surely correct in relying on the views expressed in the B.V.A. Handbook on small-animal euthanasia. To what other competent authority could they have been expected to look for guidance at that time?

Yours faithfully,

FREDERICK LAND,

8, Knighton Close,
Woodford Green,
Essex.

January 27th, 1955.

THE R.C.V.S. LIBRARY

Sir,—I was most interested to read Professor Boddie's article on the R.C.V.S. Library in to-day's issue of THE VETERINARY RECORD. During the past three years, I have had occasion to borrow several books and periodicals from the Library. I would like to record my sincere appreciation of this excellent service which is available to the profession; a service which is certainly a great asset to a practising veterinary surgeon like myself who lives so far away from Headquarters.

Yours faithfully,

ERIC I. WILLIAMS,

Trevaughan,
St. Clears, Carmarthen.

January 29th, 1955.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 31st December, 1954		25	—	—	38	—	68
Corresponding period in	1953	20	—	1	52	—	60
	1952	78	—	—	95	—	63
	1951	20	—	18	28	5	41
1st January to 31st December, 1954		350	4	12	795	—	1,455
Corresponding period in	1953	609	—	40	978	—	2,713
	1952	1,215	—	495	498	1	891
	1951	407	—	116	844	16	1,343

TUBERCULOSIS (ATTESTED HERDS) SCHEMES

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at 31st December, 1954, was as follows :—

ENGLAND	WALES	SCOTLAND	TOTAL (GREAT BRITAIN)
67,007	32,018	33,208	132,233